Science, disease and dairy production in Britain, *c.*1927 to 1980*

by Abigail Woods

Abstract

This article sheds new light on the relationship between scientific research and livestock production through a case study of the dairy cow disease, mastitis. Despite intensive scientific research, the prevalence of this widespread, costly problem barely changed in the period *c.*1927–80. Analysis of three successive framings of mastitis within the broader context of agricultural change suggests that this outcome did not reflect the failure of research, but rather its partial success. Throughout, scientists approached mastitis as a problem of production rather than health. In helping to control one form of mastitis, their investigations facilitated the adoption of more intensive farming methods, which increased milk output while encouraging the emergence of a different form of the disease. This process illustrates the co-construction of cattle health, scientific research and milk production practices. It also shows how productivist agricultural agendas and the practicalities of scientific investigation moulded the conduct of research and its effects on production.

Using bovine mastitis in Britain as a case study, this article aims to shed new light on the relationship between scientific research and the mid-twentieth century transformation of agriculture from a small-scale, self-sufficient enterprise into a specialized industrial business. For dairy farming, this transformation involved wholesale shifts in the ways that cows were fed, housed, milked, bred, monitored and managed. Adopting and applying the industrial values of efficiency and productivity, farmers sought to increase the volume and efficiency of milk production through specialization, economies of scale, mechanization, use of pharmaceuticals, a reduction in labour and the standardization of cow bodies, environments and management. In the process, producer numbers declined, herd sizes increased (Table 1), and the spaces, people and cows involved took on radically new appearances (Figures 1–3).[1]

These changes have been subject to contrasting historical interpretations. Economic, political and business historians portray them as inevitable outcomes of government policy and market

* Versions of this article were presented at the 2012 British Animal Studies Network 'Farm' meeting; the British Agricultural History Society's Winter Conference 2012, on science and agriculture; at a 2013 John Hopkins University workshop on the relationships between science, agriculture and the environment; and at the Centre for History and Philosophy of Science, Leeds University. The feedback received proved invaluable in shaping and improving the article. Especial thanks are due to Denise Phillips, Sharon Kingsland, David Edgerton, Dominic Berry, and two anonymous referees.

[1] John Martin, *The development of modern agriculture: British farming since 1931* (2000). For a discussion on the industrial ideal in American agriculture, see Deborah Fitzgerald, *Every farm a factory* (2003).

forces.[2] For scientist-participants, they were a part of their achievement in providing the nation with cheap food, [3] while for critics of intensive agriculture they were developments which were highly damaging to animal health, welfare and the environment.[4] All perspectives concur in awarding science and technology a crucial role in modernizing livestock production. However, they often portray that role in rather simplistic fashion, as the straightforward unfolding of discoveries and innovations that enabled the production of more food, more efficiently.

Recent work by historians of science adopts a more critical stance, asking why particular forms of scientific research were pursued at particular points in time, by whom, using what methods, and with what effects. In highlighting the changing nature of research agendas and the many factors involved, they show that there was no straightforward scientization of livestock production.[5] However, apart from Theunissen, who examines how dairy farmers' practices and cultures informed their adoption of scientifically informed breeding methods,[6] previous authors have been more concerned with tracing the history of science than the history of livestock production.[7] This article aims to do both, and to interrogate the relationship between them. Against a backdrop of changing production practices, and their political, economic and public health contexts, it will investigate how scientists' research programmes impacted on dairy production, and how the values and objectives of production informed the pursuit of scientific research.

The cattle disease, mastitis, offers a suitable case study because throughout the period

[2] Edith Whetham, *The agrarian history of England and Wales,* VIII, *1914–39* (1978), ch. 5; B. A. Holderness, *British agriculture since 1945* (1985); Martin, *Modern agriculture*; id., 'The commercialisation of British turkey production', *Rural Hist.,* 20 (2009), pp. 209–228; Andrew Godley and Bridget Williams, 'Democratizing luxury and the contentious "Invention of the Technological Chicken" in Britain', *Business Hist. Rev.,* 83 (2009), pp. 267–90.

[3] John Russell, *A history of agricultural science in Great Britain* (1966); G. W. Cooke (ed.), *Agricultural research, 1931–81* (1981); K. Blaxter and N. Robertson, *From dearth to plenty: the modern revolution in food production* (1995).

[4] Ruth Harrison, *Animal machines: the new factory farming industry* (1964); T. Clunies Ross and N. Hildyard, *The politics of industrial agriculture* (1992); Adrian Franklin, *Animals and modern cultures: a sociology of human–animal relations in modernity* (1999); Bernard Rollin, 'The ethics of agriculture: the end of true husbandry', in M. Dawkins and R. Bonney (eds), *The future of animal farming: renewing the ancient contract* (2008).

[5] W. Boyd, 'Making meat: science, technology and American poultry production', *Technology and Culture* 24 (2001), pp. 631–64; Mark Finlay, 'Hogs, antibiotics and the industrial environments of post-war agriculture', in Susan Schrepfer and Phillip Scranton (eds), *Industrializing organisms: introducing evolutionary history* (2004), pp. 237–60; Sarah Wilmot, 'From "public service" to artificial insemination: animal breeding science and reproductive research in early twentieth-century Britain', *Stud. Hist. and Phil. Biol. Biomed. Sci.*. 38 (2007), pp. 411–41; Paul Brassley, 'Cutting across nature? The history of artificial insemination in pigs in the United Kingdom', ibid., 38 (2007), pp. 442–61; Abigail Woods, 'The farm as clinic: veterinary expertise and the transformation of dairy farming, 1930–50', ibid., 38 (2007), pp. 462–87; Karen Sayer, 'Battery birds, "stimulighting" and "twilighting": the ecology of standardized poultry technology', *Hist. Technology* 28 (2008), pp. 149–68; Kendra Smith-Howard, 'Antibiotics and agricultural change: purifying milk and protecting health in the postwar era', *Agricultural Hist.,* 84 (2010), pp. 327–51; Margaret Derry, *Art and science in breeding: creating better chickens* (2011).

[6] Bert Theunissen, 'Breeding without Mendelism: theory and practice of dairy cattle breeding in the Netherlands, 1900–1950', *J. Hist. Biology,* 41 (2008), pp. 637–76; id., 'Breeding for nobility or for production? Cultures of dairy cattle breeding in the Netherlands, 1945–1995', *ISIS,* 103 (2012), pp. 278–309.

[7] Edgerton argues that this orientation is typical of current approaches to the history of science. David Edgerton, 'Time, money and history', *ISIS,* 103 (2012), pp. 316–27.

under review it was widely recognized as one of the most prevalent and costly problems of milk production. Generally defined as inflammation of the udder, its effects ranged from overt clinical symptoms that occasionally resulted in death, to sub-clinical reductions in the milk yield.[8] It was also believed to impact on human health, not least through the large-scale use of antibiotics to control it in the years after the Second World War.[9] Managing mastitis was the farmers' responsibility: the state played little direct role other than to fund research, most notably at the government's Central Veterinary Laboratory (CVL), Weybridge, and the National Institute for Research in Dairying (NIRD), Reading.[10] Against a backdrop of changing production practices, this account focuses on the activities, motivations and achievements of the scientists involved. While farmers' personal experiences also merit analysis, they are beyond the scope of the article.[11]

Despite intensive scientific research that produced multiple control strategies, mastitis prevalence barely changed over time. In 1934, 30 per cent of British cattle were infected, and in 1980 one third of cattle were. Today, mastitis is still the most widespread, costly disease of dairy cattle, with around 40 per cent of cows experiencing symptoms each year.[12] In order to understand this apparent failure of research, the first half of the article takes a closer look at how mastitis was conceptualized and managed from 1927, when investigations began, to 1980. Drawing on scientists' published articles and conference presentations it reveals that mastitis was not a monolithic entity during this period, but rather three different diseases, whose prevalence and perception was shaped by the politics, economics and practices of milk production.

On the basis of these findings, the next section reassesses the achievements of mastitis research and interrogates scientists' approaches to its control. It argues that, when viewed from the perspective of production rather than health, research did not fail. Rather, it facilitated the shift to more intensive systems that enhanced milk output while simultaneously encouraging the emergence of a different form of mastitis. This outcome, and the consistent manner in which scientists approached mastitis as a problem of germs rather than flawed farming systems, suggests that not only was production scientized, but also that science had been 'productivized', or constituted by the values and objectives of modern, productivity-oriented agriculture. However, as the last section of the article reveals, 'productivization' cannot fully

[8] Andrew J. Bradley, 'Bovine mastitis: an evolving disease', *Veterinary J.* 163 (2002), 1–13.

[9] The connection between bacterial resistance, as a public health problem, and the use of antibiotics in agriculture, has been hotly disputed for many decades. Smith-Howard, 'Antibiotics'; Robert Bud, *Penicillin: triumph and tragedy* (2007), ch. 8.

[10] For the history of these institutions, see H. E. Burgess, *The National Institute for Research in Dairying: a memoir* (1962); Anon, *Animal health, a centenary, 1865–1965* (1965), ch. 5. A number of other research institutions and pharmaceutical companies also conducted extensive research.

[11] Such experiences would be useful in revealing how farmers perceived and coped with the everyday, endemic diseases of livestock production. This would offer an important point of comparison with the more studied, scheduled diseases, brucellosis and tuberculosis, whose perception and management was moulded by government policy.

[12] BPP, 1933–4, cmd 4591, IX, Economic Advisory Council Committee on cattle diseases: Report, p. 427; C. D. Wilson, 'A survey of mastitis in the British dairy herd', *Veterinary Rec.* (hereafter *VR*), 106 (1980), pp. 431–5; A. J. Bradley, K. A. Leach, J. E. Breen, L. E. Green, M. J. Green, 'Survey of the incidence and aetiology of mastitis on dairy farms in England and Wales', *VR* 160 (2007), pp. 253–8.

account for the history of mastitis research because scientists recognized numerous routes to achieving higher milk output. Therefore in order to understand the historical co-evolution of science and agriculture it is necessary to consider scientists as active agents, and to scrutinize and explain their choices between different research programmes and interventions.

I

At the start of the twentieth century, mastitis was already well known to vets and farmers. It was experienced as a clinical problem of individual cows that caused sore, swollen udders and bloody milk. A bacterial cause – *Streptococcus agalactiae* – had been identified in 1887 but various predisposing factors were also thought to be involved, including poor milking technique, bad hygiene, chilling or injury to the udder, flies, and hot summer weather. Affected cows were given purgatives to treat systemic symptoms, while fomentations, stripping and massage were applied to the udder, with the occasional amputation of diseased tissue.[13]

Mastitis first attracted the attention of British scientists during the inter-war period. This was largely due to changes in dairy production. Traditionally, dairy cows formed one component of a mixed farming system, valued as much for their fertilizing manure as for their milk. However, during the inter-war agricultural depression, which impacted particularly on arable prices, producers increasingly converted to specialist dairy farming because liquid milk (although not butter and cheese) was relatively protected from foreign competition. The resulting glut led to a fall in wholesale prices which was corrected by the creation of the Milk Marketing Board (MMB) in 1993. In establishing the collective selling of milk at the best possible prices, the MMB encouraged the further expansion of dairying. By 1939, milk and its products accounted for more than 25 per cent of the value of British agricultural produce. There were over 136,000 registered producers, with herds averaging 15 cows. Each cow produced an average of 560 gallons of milk a year (see Table 1).[14]

The rising popularity and economic importance of dairy farming meant that greater significance was awarded to the diseases that threatened it. At the same time, new production practices increased the actual prevalence of disease.[15] Low arable prices enabled cattle to be fed cheaply on purchased concentrates often derived from imports. This encouraged the growth of production in urban or suburban areas where there was little grazing land but a ready market for 'producer retailers' who comprised nearly half of all producers by 1939.[16] Cows of various breed types were kept tethered in traditional sheds where they were fed and milked (Figure 1). In

[13] William Youatt, *Cattle, their breeds, diseases and management* (1858), pp. 553–4; Francis Clater, *Every man his own horse and cow doctor* (1919), revised by D. McTaggart, pp. 109–10; W. Lyle Stewart, 'Summer mastitis in cattle', *VR* 28 (1926), pp. 1055–60; S. J. Edwards, 'The diseases of dairy cattle', *J. Dairy Research,* 7 (1936), pp. 291–318.

[14] R. G. White, 'Systems of dairy farming', *J. Ministry of Agriculture,* 46 (1939–40), pp. 372–78; F. Garner, *British dairying* (1946), ch. 12; Anon, *The work of the Milk Marketing Board of England and Wales, 1933–1951* (1951); Peter Atkins, 'Fattening children or fattening farmers? School milk in Britain, 1921–41', *EcHR* 58 (2005), pp. 57–78; Paul Brassley, 'British farming between the wars', in P. Brassley, J. Burchardt and L. Thompson (eds), *The English countryside between the wars: regeneration or decline?* (2006), pp. 187–99.

[15] Woods, 'Farm as clinic'.

[16] White, 'Dairy farming'; Anon, *Milk Marketing Board.*

TABLE 1. Dairy cow statistics, 1939–85

Year	*Dairy cow numbers (England and Wales)*	*Number of registered producers (England and Wales)*	*Gross yield per cow (gallons per year) (UK)*	*Average size of herd*
1938–9	?	136,519	560	15 (est.)
1950	?	161,937	623	?
1955	2,415,000	142,792	675	17
1960	2,595,000	123,137	735	21
1965	2,650,000	100,448	780	26
1970	2,714,000	80,265	825	33
1975–6	2,701,000	59,740	902	45
1980–1	2,627,000	47,169	1037	56
1985–6	2,583,000	41,055	1078	63

Source: S. Baker, *Milk to market* (1973), p. 42; H. F. Marks and D. K. Britton, *A hundred years of British food and farming: a statistical survey* (1989), p. 230.

the absence of breeding facilities, these 'flying herds' were sustained by intermittent purchases of freshly calved cows, which were sold on when their milk output dropped. The frequent purchase and sale of cattle facilitated the spread of infection. Critics claimed that 'forcing' cows to produce in these 'intensive' or 'factory-style' systems undermined their constitutions and predisposed them to disease.[17] Outbreaks of mastitis were also associated with the adoption of milking machines, 28,860 of which had been installed on farms in England and Wales by 1942.[18] The first surveys of dairy cow health, which were brought together in a 1934 report on cattle diseases, revealed that the average cow remained in the herd for only half of her useful life. Mastitis, which infected an estimated 30 per cent of the nation's dairy cows, was the disease responsible for the greatest aggregate loss.[19]

Two of the nation's leading agricultural societies, the Royal Agricultural Society and the Royal Association of British Dairy Farmers, grew extremely concerned about the mastitis problem. Following their existing tradition of supporting scientific research,[20] they made funds available for its investigation. Additional support was provided by the Development Commission, which distributed government funds for agricultural research.[21] There were also public health reasons for conducting enquiries.[22] Ensuring the quality and safety of milk was

[17] 'Utility and endurance of dairy cows', *The Times*, 1 Feb. 1929, p. 18; S. Burdett-Coutts, 'Correspondence', *The Times*, 21 Nov. 1928, p. 20; W. C. Miller, 'Agricultural rationalism and veterinary science', *VR* 50 (1938), pp. 167–77.

[18] MAFF, *A century of agricultural statistics* (1968), p. 71.

[19] BPP, Committee on cattle diseases.

[20] Abigail Woods, '"Partnership" in action: contagious abortion and the governance of livestock disease in Britain, 1885–1921', *Minerva* 47 (2009), pp. 195–216.

[21] Nineteenth Report of the Development Commissioners for the year ended the 31st March 1929 (1929), pp. 89–90.

[22] F. C. Minett, 'Bovine streptococcus mastitis and the public health', *VR* 44 (1932), pp. 543–8. This article was published simultaneously in the journal *Public Health.*

FIGURE 1. A dairy farm in London, 1947.

This was one of the few such farms to survive the war. It exhibits many of the key characteristics of inter-war, urban or suburban dairy farming.
Farmer and Stockbreeder Collection. Reproduced with the permission of the Museum of English Rural Life, University of Reading.

a high-profile political issue in the inter-war period.[23] Although the key focus of concern was bovine tuberculosis, mastitis also attracted attention. Most commonly it caused rapid souring and visible changes in the colour and consistency of milk, but in addition, between 1903 and 1934, mastitis-infected milk was associated with over 100 outbreaks of human epidemic disease.[24] An aetiological connection was suggested just prior to World War I by the discovery in the throats of patients with scarlet fever and septic sore throat, of a streptococcus bacterium similar to that associated with mastitis in cows.[25]

Veterinary bacteriologists at the Royal Veterinary College, London, sought to elucidate this problem. Building on prior investigations in the USA, Frank Minett and his assistants

[23] Frank Trentmann, 'Bread, milk and democracy: consumption and citizenship in twentieth-century Britain', in M. Daunton and M. Hilton (eds), *The politics of consumption* (2001), pp. 129–63; M. French and J. Phillips, *Cheated not poisoned? Food regulation in the United Kingdom, 1875–1938* (2000), ch. 7.

[24] BPP, Committee on cattle diseases, pp. 28, 89.

[25] 'Discussion on milk borne streptococcus epidemics', *Proc. Royal Society of Medicine*, 24 (1931), pp. 1703–12; L. Wilson, 'The historical riddle of milk-borne scarlet fever', *Bull. of the Hist. of Medicine* 60 (1986), pp. 321–42.

A. J. Stableforth and S. J. Edwards used classic 'microbe hunting' techniques to discover a range of bacteria capable of causing mastitis, and to dispel fears that they could infect humans. Subsequently, they chose to focus on the bacterium implicated in 80 per cent of mastitis cases: *Streptococcus agalactiae*. This lived mainly on the udder skin, spread via milking, and caused infection by migrating up the teat canal.[26] Unlike vets and farmers, scientists were not concerned with the clinical cure of individual animals, but with the herd-level manifestations and control of disease. They claimed that while most stockmen were aware of the acute form of mastitis, it was more usually chronic in nature. Manifesting not in symptoms but in reduced milk yield, the only reliable method of detecting it was through the bacterial culture of milk.[27] Since infection occurred independently in each quarter of the udder, samples had to be taken from each teat. This approach made the laboratory an obligatory passage point in mastitis diagnosis, and reduced the individual cow to a collection of udder 'quarters'.[28]

The standard response to infectious cattle diseases in the 1930s was to try to eradicate them from the herd, if not the nation. Drawing on methods already applied to bovine tuberculosis and brucellosis,[29] Minett and his colleagues advocated bacterial testing to identify infected cows, which could then be sold or separated off into a different herd and milked last. Follow-up tests were required to check infection status. As an adjunct therapy, the udders of diseased cows could be infused with a bactericidal agent like acriflavine, although this temporarily reduced milk output and often failed to eliminate infection. Mastitis vaccines and sera, which drug companies included in their growing range of biological products, had little demonstrable effect.[30]

In the later 1930s, supported by the newly established, government-funded Agricultural Research Council (which regarded animal disease research as a major priority) and the MMB (which was concerned by the losses caused by mastitis), scientists embarked on field trials of their herd eradication plan. The ARC set up a technical committee to co-ordinate this work, which took place at the RVC, the Hannah research institute in Ayrshire where S. J. Edwards had moved in 1934, and at two Scottish agricultural colleges.[31] Results were disappointing. This was explained later by the discovery of carrier cows that were infected but did not always shed bacteria in milk.[32]

[26] F. C. Minett, A. W. Stableforth and S. J. Edwards, 'Studies on bovine mastitis: i) The bacteriology of mastitis', *J. Comparative Pathology and Therapeutics* 42 (1929), pp. 213–31; E. Munch Peterson, *Bovine mastitis. Survey of the literature to the end of 1935* (1938).

[27] F. C. Minett and W. J. Martin, 'Influence of mastitis and brucella abortus infection upon the milk yield of cows', *J. Dairy Research* 7 (1936), pp. 122–44.

[28] F. C. Minett, A. W. Stableforth, S. J. Edwards, 'Studies on bovine mastitis: iii) The diagnosis of streptococcus mastitis', *J. Comp. Path. Ther.,* 43 (1930), pp. 163–88; Edwards, 'Diseases', pp. 291–99.

[29] Woods, 'Partnership'; Keir Waddington, 'To stamp out "so terrible a malady": bovine tuberculosis and tuberculin testing in Britain, 1890–1939', *Medical Hist.,* 48 (2004), pp. 29–49.

[30] A. W. Stableforth, 'Mastitis in dairy cows and its control', *J. Min. Ag.,* 41 (1934–5), pp. 945–55; Edwards, 'Diseases'; A. W. Stableforth and N. Scorgie, 'Entozon and acriflavine for the treatment of chronic contagious bovine mastitis', *VR* 50 (1938), pp. 661–76.

[31] TNA, JV 10/257, Report of conference on mastitis research, 20 May 1938 and ARC committee on mastitis: Report for the period ended 31 Dec. 1940; Timothy DeJager, 'Pure science and practical interests: the origins of the Agricultural Research Council, 1930–37', *Minerva,* 31 (1993), pp. 129–37.

[32] Agricultural Research Council, *Modes of spread of Streptococcus agalactiae infection in dairy herds* (1944).

In wartime, patterns of dairy farming shifted to more extensive production, as scarce shipping space and the loss of shipping to enemy action in the Atlantic reduced the supply of imported concentrate feed. While these changes constrained the keeping – and detrimental health impacts – of 'flying herds', labour shortages caused farmers to turn increasingly to machine milking, which was applied to one third of the cows in Britain by the end of the war. Due to the nutritional importance of milk and the capacity for its domestic production, dairy farming was actively encouraged by government. Producers received generous set prices and expert guidance on how to grow fodder crops.[33]

This effort to increase milk production resulted in the intensification of state-supported mastitis research. Multiple institutions conducted enquiries, including the government's Central Veterinary Laboratory (CVL), Weybridge, where A. W. Stableforth now headed the bacteriology department; the ARC's field station at Compton, where S. J. Edwards moved in 1939; the Hannah Institute; the East and West of Scotland agricultural colleges; the National Institute of Research in Dairying (NIRD), Reading; the Dick (Edinburgh) veterinary school and the Moredun Institute. Privately funded enquiries were carried out in the research laboratories of Boots and Burroughs Wellcome drug companies. Scientists met to report on their progress and future plans at an annual ARC 'mastitis conference' chaired by the director of the CVL, Thomas Dalling.[34]

Shortages of glassware, laboratory and advisory staff impeded the wartime continuation of inter-war bacterial testing for mastitis.[35] Instead, control was pursued under a clinically based scheme, proposed by the National Veterinary Medical Association and implemented by practising vets, who diagnosed mastitis on clinical grounds. Infected cows were segregated, milked last, and their udders infused with an anti-bacterial agent, acriflavine. Systemic symptoms were treated with government-subsidized sulphanilamide.[36] These measures were not particularly successful.[37] However, the development of penicillin offered scientists a new tool for achieving their existing goal of herd eradication. Due to the political and nutritional importance of milk production they were granted preferential access to this scarce drug. Mastitis therapy trials began in 1943 and gave promising results.[38]

Mastitis research remained a priority after the war as food shortages continued and producers strove to raise output. In 1945, scientists embarked on large-scale field trials of penicillin therapy in commercial herds located near the CVL (where A. W. Stableforth replaced Dalling as director in 1950), Compton and NIRD. Similar efforts were undertaken by the drug companies, Burroughs Wellcome and Boots. Researchers continued to share their findings at the annual mastitis conference. From working with the same herds over a number of years, they developed an understanding of the practices, priorities and psychology of farmers and

[33] Martin, *Modern agriculture*; Woods, 'Farm as clinic'.

[34] TNA, MAF 52/257, Diseases of dairy cattle: memo to milk production policy committee, April 1940; MAF 189/434, ARC conferences on mastitis, 1944.

[35] TNA, MAF 35/488, Notes on a scheme formulated by the mastitis committee of the ARC, 1940 and Minutes of meeting on livestock disease control, 20 Dec 1940.

[36] NVMA, 'Report on diseases of farm livestock', *VR* 53 (1941), pp. 3–14. For details of the scheme, which also included infertility, see Woods, 'Farm as clinic'.

[37] P. S. Watts, 'The practitioner and mastitis control under the national scheme', *VR* 55 (1943), pp. 175–9.

[38] S. J. Edwards and A. Brownlee, 'Therapeutic treatment of bovine mastitis', *VR* 55 (1943), pp. 335–43.

their workers. These insights guided their search for the simplest, cheapest, most effective penicillin preparations and dose regimes.[39]

Penicillin trials reasserted the bacteriological approach to mastitis control. Laboratory tests were used to identify cows infected with *Str. agalatiae* and to determine the effects of treating them with repeated doses of penicillin, a regime that became known as 'blitz therapy'. Drug companies facilitated these efforts by devising, in collaboration with field-based researchers, a straightforward method of administering penicillin via single-use intra-mammary tubes.[40] However their influence should not be overestimated because drug-centred, bacteriological approaches to mastitis pre-dated their involvement. Herd trials continued until 1953, by which time penicillin had been released for use by practising vets and was proving its worth.[41] In 1950–52, *Str. agalactiae* caused less than ten per cent of mastitis cases, compared to 80 per cent in the 1930s. Incidence had dropped to four per cent by 1956, and hundreds of herds were freed entirely of infection.[42]

This brief account shows that mastitis research in Britain was initially dominated by veterinary bacteriologists, who defined the disease as the product of udder invasion by *Str. agalactiae* bacteria. Unlike farmers and practising vets, they were not concerned with the fate of individual animals or with achieving clinical cure. Although production practices were widely associated with the mastitis problem, they did not attempt to investigate their contribution. Approaching mastitis as a bacterial infection of the herd, their preferred solution was to detect germs by bacteriological testing, and then eliminate them, initially by hygienic and later by pharmaceutical means.

II

Although *Str. agalactiae* incidence had reduced considerably by the 1950s, there was no reduction in the clinical and sub-clinical forms of mastitis caused by a different germ, *Staphylococcus aureus*. In fact its prevalence appeared to be increasing. The habitat of *Staph. aureus* was not restricted to the udder, allowing it to evade the effects of penicillin therapy.[43] The causes of increased prevalence provoked considerable debate. Some asked whether in eradicating *Str. agalactiae*, penicillin had somehow facilitated the emergence of *Staph. aureus*.[44] Others pointed to post-war changes in breeding and husbandry practices, which were prompted by government efforts under the 1947 Agriculture Act to bring stability to agriculture, and encourage expansion of output in the face of continuing food shortages.

[39] TNA MAF 189/434–6, Mastitis conference papers, 1944–47.

[40] Ibid., also A. W. Stableforth, 'Penicillin in str agalactiae infection: trials made in Britain in 1945 and 1946', *VR* 61 (1949), pp. 235–6.

[41] ARC Technical Committee on Mastitis, 'A method for the eradication of strep agalactiae', *VR* 67 (1955), pp. 410–11.

[42] MAFF, *Machine Milking* (Bulletin no 177, 1959), pp. 105–6.

[43] J. Berger and J. Francis, 'Mastitis in practice: epidemiology and bacteriology', *VR* 63 (1951), pp. 283–92; TNA MAF 189/664, Reports by D. L. Hughes and J. L. K. Pearson to mastitis conferences, 1952, MAF 189/665; D. Howell and C. D. Wilson, reports to mastitis conference, 1955.

[44] 'Report: management factors in the control of mastitis', *VR* 63 (1951) p. 211; C. D. Wilson, 'Factors that predispose to mastitis with special reference to milking technique', *VR* 70 (1958), pp. 159–66.

FIGURE 2. Milking pits and tandem units, 1956.

Farmer and Stockbreeder Collection. Reproduced with the permission of the Museum of English Rural Life, University of Reading.

In addition to an assured market and guaranteed prices, the 1947 Act offered increased access to capital grants. Some producers used these to erect separate hygienic dairies for machine milking (Figure 2) and labour-saving loose housing (Figure 3) where cows could exercise freely and be fed silage en masse. Higher yielding, specialist milk breeds, particularly Friesians (as in Figure 3) were adopted in place of dual purpose cows that could be used for milk or meat, while new artificial insemination services run by the Milk Marketing Board allowed producers to use proven bulls to father more productive offspring. The 1953 relaxation of controls on purchased feed encouraged a shift to pre-prepared rations, devised by feed companies on the basis of new scientific research. Together with the state-led control of bovine tuberculosis, and the mainstream application of antibiotics, these changes caused the national output of milk to rise 131 per cent above the pre-war average by 1952–3, while the milk output per cow rose from 560 to 675 gallons between 1939 and 1955 (Table 1).[45]

[45] Paragraph drawn from K. Russell, *The principles of dairy farming* (1953), ch. 1; Anon, *Animal health*; S. Baker, *Milk to market: forty years of milk marketing* (1973); Martin, *Modern agriculture*; Woods, 'Farm as clinic'; T. Corley and A. Godley, 'The veterinary medicines industry in Britain, 1900–2000', *EcHR* 64 (2011), pp. 832–54.

FIGURE 3. Cows on slatted floors, 1961.

Farmer and Stockbreeder Collection. Reproduced with the permission of the Museum of English Rural Life, University of Reading.

Commentators, who included some mastitis researchers, suggested that these 'unnatural' changes in breeding and husbandry, and the continuing uptake of machine milking, had upset the proper balance between animals, microbes and their environment.[46] They argued that selection for high milk yields had made cows less resistant to mastitis infection, and produced overdeveloped udders that were more susceptible to injury from slatted floors and faulty milking machines.[47] In this interpretation, bacterial mastitis was secondary to bad husbandry.[48]

Husbandry changes continued to accelerate in the next decade, as the near-achievement of national self-sufficiency in milk production caused the government to reduce its subsidies. In the 15 years from 1954/5 over 60,000 farmers chose to leave milk production, but those who remained were forced to become more efficient in order to survive. The search for economies of scale caused average herd size to rise to 33 cows by 1969–70, with no corresponding increase

[46] C. D. Wilson, 'Field control of mastitis', *State Veterinary J.*, 9 (26) (1954), p. 22.

[47] Wilson, 'Factors that predispose'; C. D. Wilson, 'Man, machines and mastitis', *VR* 75 (1963), p. 1312.

[48] TNA, MAF 189/666, Minutes of Scottish AIC committee on mastitis, 29 Oct 1958; MAF 189/666, J. W. Howie, correspondence to A.W. Stableforth, 19 Nov 1962. For a critique of this view, see F. H. Dodd, F. K. Neave, R.G. Kingwill, 'Control of udder infection by management', *J. Dairy Sci.*, 47 (1964), 1109–14.

in labour. The time available to tend to each cow fell from 124 to 88 hours per worker per year. Machine milking became almost universal, and some producers adopted new parlour systems like the herringbone in an attempt to use labour more efficiently. Ongoing selection for milk production and the continuing shift away from dual purpose to specialist milk breeds (Friesians made up 64.2 per cent of the national herd in 1965, compared to 40.6 per cent in 1955) caused output to increase to 780 gallons per cow per year by 1965. Milk was increasingly handled in bulk tanks rather than churns, and different forms of labour-saving cow housing emerged, including cow cubicles. This period also saw a growth in farm management techniques and services, as farmers sought to quantify their inputs and outputs, and rationalize their businesses.[49]

A series of disease surveys conducted in the late 1950s and early 1960s revealed the scale of the mastitis problem. Ten per cent of cows experienced clinical mastitis each year, of which 30 per cent was caused by *Staph. aureus*. Around 25 per cent of cows had sub-clinical mastitis at any point in time. This caused a 10 per cent drop in milk yield and an 11 per cent drop in solids-not-fat content, a measure that now influenced the price paid for milk.[50] Bacterial resistance to antibiotic therapy was increasing, and streptococcal infection soon returned to pre-penicillin levels. The leading veterinary expert on *Staph. aureus* mastitis, C. D. Wilson, voiced concerns that 'we shall be doomed to try out, use, and then discard one antibiotic after other until cows will be infected with some of the most resistant staphylococci known to man'.[51] Despite growing recognition of the limits to antibiotic therapy, its ease of administration meant that it remained popular with farmers and practising vets, to the extent that in 1963, 11 per cent of milk samples were found to contain antibiotic residues. This not only created problems for milk processors reliant on bacterial cultures for the manufacture of cheese. It also posed a new threat to the public's health. Contaminated milk endangered humans allergic to penicillin, and concerns emerged that antibiotic use in agriculture might promote resistance amongst human pathogens.[52]

Drug companies responded to this situation by seeking alternative anti-bacterial agents, while scientists at the ARC's Compton laboratory engaged in an ultimately fruitless search for vaccines.[53] A different approach was adopted by dairy bacteriologists and husbandry experts at the NIRD, including F. H. Dodd, F. K. Neave and R. Kingwill. Departing from the existing veterinary paradigm of germ-focused enquiries, they proposed, in 1949, to study the 'soil' as well as the 'seed' of mastitis. Whereas penicillin use had partially concealed the health impacts of changing dairy husbandry, they aimed to make these effects visible by approaching mastitis as a problem of management. They proposed to use identical twins to establish two

[49] Martin, *Modern agriculture*; G. F. Smith, 'Evaluating recent movements in the dairy industry', *VR* 84 (1969), pp. 388–90; Baker, *Milk to market*; K. Russell, *The principles of dairy farming* (seventh edn, 1974); O. Grant, 'The diffusion of the herringbone parlour' (University of Oxford, discussion paper in economic and social history, 1998).

[50] C. D. Wilson, 'The treatment of staphylococcal mastitis', *VR* 73 (1961), pp. 1019–20; D. Howell, C. D. Wilson and M. P. Vessey, 'A survey of the incidence of mastitis in dairy cows', *VR* 76 (1964), pp. 1107–12.

[51] Wilson, 'Staphylococcal mastitis'.

[52] Milk and Milk Products Technical Advisory Committee, *Report on antibiotics in milk in Great Britain* (1963); Bud, *Penicillin*, pp. 175–77.

[53] J. Carmichael *et al.*, 'The therapy of penicillin resistant mastitis', *VR* 62 (1950), pp. 55–8; C. D. Wilson, 'The present position with regard to bovine mastitis and a review of recent trials of various antibiotics', *VR* 66 (1954), pp. 775–88.

separate herds which could be kept under controlled conditions. The effects of different factors on cow susceptibility to mastitis would then be studied in turn.[54] Meanwhile, at the CVL, C. D. Wilson tested new antibiotic products, and worked with another vet, I. Davidson, on the 'ecology of mastitis staphylococci'. This involved bacteriological and epidemiological research into the sources of *Staph. aureus* and how milking influenced its spread.[55]

Drawing on wider epidemiological thinking,[56] knowledge of *Staph. aureus* epidemiology, and insights gained from working closely with dairy farmers, Wilson and the NIRD scientists developed a shared perspective on mastitis. Agreeing that 'straightforward infection isn't the whole story',[57] they saw it as the outcome of dynamic herd-level interactions between populations of germ parasites and their udder hosts. It required a 'stress factor' or 'trigger mechanism' to disturb the normal state of balance between host and parasite,[58] with disease occurring when 'sufficiently heavy infection is brought to sufficiently susceptible animals under conditions that will favour establishment of the infection in the bovine udder'.[59] Milking was heavily implicated, both in transferring germs between cows and inflicting mechanical damage on the teats, thereby enabling germs to invade. Correspondingly, scientists' investigations focused on the design, operation and use of the milking machine.[60]

This approach was less reductionist than of the earlier period. It did not focus simply on germs but also the milking process. Instead of eliminating germs from cows it sought their peaceful co-existence. However, curing the individual sick cow, and elucidating the causal role of wider management factors were again neglected as scientists continued to focus on herd health and production.[61] Already favoured by the growth in herd size and reduction in labour, this perspective was facilitated by a new method of detecting udder inflammation from the number of cells shed in milk. Devised in 1957, the California Milk Test (CMT) made sub-clinical mastitis visible. The results correlated with bacteriological testing and were considerably easier and cheaper to obtain. When applied to the bulk milk (the bulk milk cell count, BMCC), it indicated the mastitis status of the herd.[62]

In devising methods of controlling *Staph. aureus* mastitis, scientists kept the needs and perspectives of dairy farmers in mind, and sought ways of encouraging and empowering them

[54] TNA MAF 189/358, Mastitis conference meeting, 6 Dec. 1949.

[55] Wilson, 'Field control'; I. Davidson, 'The ecology of mastitis staphylococci', *State Veterinary J.,* 13 (39) (1958), pp. 36–40; Wilson, 'Staphylococcal mastitis'.

[56] R. Lovell, 'Epidemiology', *British Veterinary J.,* 121 (1965), pp. 421–6.

[57] TNA, JV 10/257, ARC conference on mastitis, 7 Oct. 1966.

[58] Wilson, 'Factors that predispose'; Dodd, Neave and Kingwill, 'Udder infection'.

[59] C. D. Wilson and I. Davidson, 'The control of staphylococcal mastitis', *VR* 73 (1961), p. 321.

[60] On udder management, see the papers by J. Oliver, F. H. Dodd and F. K. Neave in *J. Dairy Res.,* 23 (2) (1956). Many aspects of machine milking were researched at the NIRD during this period. See MAFF, *Machine milking.* On its relationship to mastitis, see F. H. Dodd and F. K. Neave, 'Milking machine rate and mastitis', *J. Dairy Res.,* 18 (1951), pp. 240–45; F. H. Dodd, J. Oliver and F. K. Neave, 'The effect of design of teat cup liners on mastitis incidence', *J. Dairy Res.,* 24 (1957), pp. 20–26.

[61] For the influence of these management factors on mastitis, see the document drawn up by the British Veterinary Association's Technical Development Committee: 'Controlling bovine mastitis', *VR* 77 (1965), pp. 612–22.

[62] Scottish mastitis researchers applied cell counting prior to the development of the CMT, but their methods were not widely accepted. TNA MAF 189/665, Minutes of mastitis conference, 26 Oct. 1955 and 21 June 1956. Blackburn reviewed the literature on the CMT in 'Reviews of the progress of dairy science', *J. Dairy Res.,* 25 (1958), pp. 494–5.

to act. Recognizing the limited time and money available for mastitis control, they sought simple, proven, cost-effective controls that would work in all production systems and against all germs.[63] Initially they focused on the proper use of the milking machine. In 1966, following promising experimental results, they began field trials in commercial herds of a simple, quick hygienic milking routine.[64] Although this reduced mastitis levels, scientists felt that farmers would be discouraged by its slow rate of progress, and so decided to incorporate antibiotics for treatment of clinical cases and of all cows at drying off. They also recommended regular cell counts, on the grounds that they would make mastitis visible, demonstrate herd-level effects of interventions, and thereby inspire and maintain farmers' interest in mastitis control.[65]

From 1968, these measures were piloted by a new Mastitis Research Unit established by the MMB, whose findings guided the development of a voluntary nationwide BMCC service.[66] In 1972, controls were codified and advertised to farmers and practising vets under a Mastitis Awareness Campaign, launched jointly by all organizations with an interest in milk production (including the National Farmers' Union, MMB, British Veterinary Association and pharmaceutical companies). This identified six action points. The first, good management, had not been subjected to scientific investigation, and was defined in vague terms as 'good housing, feeding and adequate bedding.' It was later dropped to produce what became known as the 'five point plan':[67] teat dipping in disinfectant after milking; prompt antibiotic treatment of clinical cases; antibiotic treatment of all cows when milking ceased before calving (known as 'dry cow therapy'); culling of chronically infected cows; annual testing of milking machines, and monthly bulk milk cell counts.[68]

Elements of this plan were adopted unevenly by livestock producers, and it was not an immediate success.[69] Slowly, however, in the 15 years from 1966, the national BMCC fell from over 600,000 to just over 400,000. The proportion of infected cows dropped from 50 per cent to 32 per cent, and the number of clinical cases from 150 to 70 per 100 cows per year. The plan was also taken up internationally, and widely regarded as an economic success.[70] High prices

[63] Dodd, Neave and Kingwill, 'Udder infection'; F. K. Neave, F. H. Dodd and R. Kingwill, 'A method of controlling udder disease', *VR* 78 (1966), pp. 521–3; R. G. Kingwill, F. K. Neave, F. H. Dodd, T. K. Griffin, D. R. Westgarth, C. D. Wilson, 'The effect of a mastitis control system on levels of subclinical and clinical mastitis in two years', *VR* 87 (1970), pp. 94–100.

[64] Similar measures were tested concurrently at Cornell University veterinary school. Kingwill *et al*., 'Mastitis control system'; C. D. Wilson and R. G. Kingwill, 'A practical mastitis control scheme', *International Dairy Federation Annual Bulletin,* 85 (1975), pp. 422–38.

[65] C. D. Wilson, 'The mastitis awareness campaign in the United Kingdom', *State Vet. J.,* 29 (87) (1974), pp. 153–62. BMCCs were introduced into Northern Ireland in 1968 and Scotland in 1970, and formed the basis of mastitis control programmes in other countries. The EEC proposed to link measurements to the payments received for milk. J. K. L. Pearson, D. O. Greer, B. K. Spence, 'Mastitis control: the use of electronic cell counting methods', *VR* 90 (1972), pp. 485–6; TNA, JV 10/258, MMB, 'Mastitis: a brief review of incidence and control', 25 Jul. 1968 and Scottish MMB document, Mar. 1970.

[66] TNA, JV 10/258, MMB, Review of mastitis research unit, 1968–9.

[67] M. Green and A. Bradley, 'The changing face of mastitis control', *VR* 173 (2013), p. 518.

[68] The MMB started a milking machine testing service in 1966. Statistics in 1969 showed that 88 per cent of machines had at least one major fault. 'News', *VR* 85 (1969), p. 365; Wilson, 'Mastitis awareness campaign'.

[69] D. A. K. Thornton, 'The Southern Counties Veterinary Society Mastitis Control Scheme', *VR* 90 (1972) , pp. 68–71; Editorial, 'Controlling mastitis', *VR* 93 (1973), p. 527.

[70] Wilson and Richards, 'Survey'; Bradley, 'Survey'.

for cull cows, the ongoing decline in producer numbers (those leaving the industry often had above average cell counts), the rolling out of BMCCs to all producers in 1977, NIRD research into the actual costs and benefits of mastitis control, and threatened penalties for high cell count milk (eventually introduced in 1991) all contributed to this outcome.[71]

III

Concurrently with the implementation of the NIRD/CVL mastitis plan, a new form of mastitis rose to prominence. Caused particularly by coliform bacteria (*E. coli* and *Klebsiella*) and *Streptococcus uberis*, it was associated with acute clinical symptoms, which often occurred around calving time and could end in death. Cows that survived took time to recover and their milk output rarely returned to pre-infection levels. The condition was referred to as 'environmental mastitis' because the causative agents did not live on the cow. This meant that they were little affected by milking hygiene or dry cow antibiotics.[72] Compared to the sub-clinical forms of mastitis which could only be detected by the BMCC, environmental mastitis was more visible and costly. Farmers referred cases to practising vets, whose reports to the veterinary press alerted scientists to the scale of the problem.[73] The results of bacteriological testing showed that, whereas in the early 1940s these germs were present in less than two per cent of milk samples taken from clinically affected cows, in 1960 they were found in 9.8 per cent of samples, and around 50 per cent by 1975. On some farms they were responsible for up to 80 per cent of clinical mastitis cases.[74]

Environmental mastitis often occurred on large, intensive farms, where standards of mastitis control – as defined by adherence to the CVL/NIRD plan – were high. This finding drew attention, once more, to the continuing intensification of dairy production as a contributing factor to mastitis. While Britain's 1973 entry into the EEC temporarily eased economic pressures on farming by increasing the price of milk used for manufacturing, surpluses soon emerged. Subsidies were removed entirely in 1977, and in 1984, quotas were introduced. By then, there were only around 40,000 milk producers left in England and Wales. Dairy cow numbers fell slightly as herd sizes increased to an average of 98 cows in the south of the country and 59 in the north. Rotary parlours enabled faster milking, while producer retailers virtually disappeared. Selection for yield continued, and electronic methods of identifying cows were devised, which enabled precise quantities of feed to be delivered automatically at milking time. These developments raised average annual yields to well over 1000 gallons by 1985, from a national herd comprising 95 per cent Friesian cows.[75]

[71] C. B. Asby *et al.*, 'The benefits and costs of a system of mastitis control in individual herds' (University of Reading, Department of Agriculture, study no 17, 1975); TNA, JV 10/260; J. Booth, Reports to the MMB: Mastitis, 1972–76, JV 10/261, J. Booth, 'A review of the current mastitis situation', report no 618 to MMB, Nov. 1978.

[72] Bradley, 'Survey'.

[73] Technical Development Committee, 'Coliform and pseudomonad mastitis: epidemiology and control', *VR* 100 (1977), pp. 441–2; A. Marr, 'Bovine mastitis control: a need for appraisal?', *VR* 102 (1978), pp. 132–4.

[74] D. Howell, 'Survey on mastitis caused by environmental bacteria', *VR* 90 (1972), pp. 654–7; Technical Development Committee, 'Coliform'.

[75] K. Russell, *The principles of dairy farming* (tenth edn, 1985).

Some mastitis commentators suggested that these changes in cow genetics and management had increased susceptibility to environmental mastitis,[76] and that 'the concentration of large numbers of animals, frequently held under conditions of stress, could increase the danger of cross infection'.[77] Others asked whether dry cow antibiotic therapy had killed off harmless bacteria in the teats and udder, whose presence had stimulated the production of defensive inflammatory cells. American researchers, Schalm and Lasmanis, provided support for this view by showing that a BMCC of more than 300,000 could protect the udder against coliform mastitis. Such findings cast doubt on the merits of striving for ever-lower BMCCs, and on the CVL/NIRD plan itself, with critics asking whether, in the light of recent husbandry changes, its findings were still relevant. Some even suggested that farmers abandon it, as although sub-clinical mastitis would result, this was less costly and dramatic than the environmental form.[78]

Although they were forced to admit that environmental mastitis was a growing problem, CVL and NIRD scientists were not convinced by the evidence implicating antibiotics, and continued to uphold their plan, as did veterinary representatives of drug companies like Beechams which had invested heavily in the antibiotic control of mastitis.[79] While drug companies sought new antibiotic solutions,[80] scientists at the NIRD focused, once more, on preventing the dissemination and invasion of germs. Although for farmers and practising vets, environmental mastitis was a disease of individual cows, requiring management on a case by case basis, scientists still regarded it as a herd problem and continued to neglect its predisposing causes. However, in a new departure that may have reflected the wider recognition of environmental hazard as disease risk, they extended their gaze beyond the milking process, to the cow's immediate environment.[81]

On discovering that the usual source of *E. coli* was faecal contamination of the teat, which occurred mainly between milkings, scientists looked for ways of stopping germs from gaining access. While turning cows out to grass or reducing stocking densities could have achieved this goal, they deemed these measures to be impractical. Having identified an association between mastitis incidence and the bacterial population of the cow's bedding, they attempted to reduce bacterial numbers by changing the bedding material, disinfecting it, regulating its temperature (which influenced the speed of bacterial multiplication) and cleaning out regularly.[82] While these measures helped in some herds, environmental mastitis remains a problem to this day.

[76] J. O. L. King, 'Mastitis as a production disease', *VR* 91 (1972), pp. 325–30.

[77] G. C. Brander, 'Dairy herd environment and the control of mastitis', *VR* 92 (1973), pp. 501–06.

[78] Howell, 'Survey'; Technical Development Committee, 'Coliform'; A. Marr, 'Bovine mastitis control: a need for appraisal?', and subsequent correspondence, *VR* 102 (1978), pp. 132–4, 321, 369–70 and *VR* 103 (1978), p. 39.

[79] Wilson, 'Mastitis awareness campaign'; G. B. Brander, 'Bovine mastitis control', *VR* 102 (1978), p. 221; G. H. Yeoman, 'Bovine mastitis control', *VR* 102 (1978), pp. 291–2; A. J. Bramley, 'Bovine mastitis control', *VR* 102 (1978), pp. 447–8; A. J. Bramley and F. H. Dodd, 'Reviews of the progress of dairy science: mastitis control – progress and prospects', *J. Dairy Res.,* 51 (1984), pp. 481–512.

[80] H. Watkins and J. F. Buswell, 'The treatment of clinical mastitis with a combination of ampicillin and cloxacillin', *VR* 96 (1975), pp. 289–90.

[81] L. Nash, *Inescapable ecologies: a history of environment, disease, and knowledge (*Berkeley, 2006).

[82] A. J. Bramley and F. K. Neave, 'Studies on the control of coliform mastitis in dairy cows', *British Vet. J.,* 131 (1975), pp. 160–69; Technical Development Committee, 'Coliform'; Bramley and Dodd, 'Reviews'.

IV

Our analysis reveals that although mastitis was consistently regarded an important condition that merited intensive scientific research, it was not a uniform entity during the period 1927–80, but rather three different diseases that were problematized and investigated in turn. Each involved a different bacterial cause and was associated with changing production practices, themselves moulded by the economic and political context and public health concerns. While scientists tailored their research and control strategies to each of these three diseases, their activities also reveal certain continuities. Despite acknowledging that mastitis was more of a problem within intensive systems, their published research did not attempt to find out why, or to examine how production systems could be adjusted in order to enhance cow health. Although they gradually extended their gaze from the cow, to the milking machine and then to bedding, scientists consistently constructed mastitis as a problem of bacteria rather than flawed production systems, and focused their energies on tracking and controlling the germs responsible. Individual cows (and individual farmers) were similarly overlooked in their search for universally applicable, herd-level solutions.

So what did the scientization of mastitis control achieve? When viewed in isolation, the statistics give an impression of failure: 30 per cent of British cattle were reportedly infected in 1934, and 33 per cent in 1980.[83] However, the above frame-by-frame analysis permits a more nuanced reading. Both the antibiotic blitz therapy directed against *Str. agalactiae*, and the NIRD/CVL plan to manage *Staph. aureus* achieved some success in controlling the germs targeted. However, these gains were partially concealed by corresponding increases in mastitis caused by different germs. Therefore one possible interpretation of the achievements of mastitis research is that, like the nineteenth century cropping innovations studied by Paul Olmstead and Alan Rhodes,[84] it managed to shore up cattle health in the face of ongoing threats: scientists stopped the situation from getting worse.

However, it could also be argued that scientists' partial solutions contributed to the recreation and re-problematization of mastitis. While the causes and extent of antibiotic resistance are difficult to discern, scientists may have facilitated its development by promoting the routine use of antibiotics. Antibiotic therapy also generated public health concerns about milk residues, and was blamed, in the later 1970s, for increasing cow susceptibility to environmental mastitis.[85] In addition, by helping to tackle a disease that was already more of a problem in productivity oriented systems, scientists enabled producers to continue reshaping cow bodies and environments without worrying about the limits imposed by the disease. They thereby helped to create conditions conducive to the emergence of a different form of mastitis, which then became the subject of renewed scientific research. In this way, mastitis research, production practices and cow health were co-constructed.

In this process of co-construction, the scientization of livestock production was coupled with

[83] Bradley, 'Survey'.

[84] A. Olmstead and P. Rhodes, 'Biological innovation in American wheat production: science, policy and environmental adaptation', in S. Schrepfer and P. Scranton (eds), *Industrialising organisms* (2004), p. 70.

[85] Bramley and Dodd, 'Reviews'.

the 'productivization' of science. Internalizing the goals and values of productivity-oriented dairy farming, mastitis researchers aimed not to improve cow health as an end in itself, but rather to tackle its detrimental effects on milk production. From the outset, their research was inspired by the high prevalence of infection, which was mostly sub-clinical and therefore evident only in reduced milk output. Managing the highly visible and costly effects of mastitis in individual animals was left to the practising veterinary surgeon. Public health concerns also stimulated research, but mainly by alerting agricultural interests to the prospect of reduced consumption of, and confidence in milk, to the detriment of farming profits. Public health experts did not participate in mastitis enquiries. The key figures were veterinary bacteriologists and dairy husbandry experts, who were oriented towards farmers and the state. Their field trials brought them closely into contact with producers, who were under considerable pressure to increase the volume and efficiency of milk output. Such pressure was exerted by market forces, which the government manipulated during and after the Second World War through its subsidy regime, while simultaneously funding research that could help farmers to achieve these ends. In accordance with the goals of their government funders and farming audiences, scientists tailored their mastitis investigations to meet the objectives of modern, intensive production.

This finding suggests a further interpretation of the achievements of mastitis research: scientists' partial solutions actually succeeded because, during the period under review, milk production per cow doubled in spite of the continuing prevalence of mastitis (Table 1). Of course mastitis control was not the only factor responsible for increased productivity. Many other innovations occurred concurrently. But, in tackling a disease that was known to be more of a problem in intensive systems, scientists helped these systems to survive and progress, thereby facilitating the pursuit of productive efficiency.

V

The 'productivization' of mastitis research helps to explain why scientists approached it in the ways that they did, and to resolve the apparent paradox whereby they recognized the role of intensive systems in producing mastitis, yet ignored such systems in their research. When viewed from the perspective of production, their construction of mastitis as a herd-based problem rather than a disease of individual cows makes perfect sense because cumulatively, its sub-clinical effects on herd milk output were more costly than its individual clinical effects. Inspired by the standardization of cow bodies and environments within intensive systems, scientists sought standardized methods of mastitis control that would prove both practical and profitable to the farmer. Realizing that changes to the farming system might reduce mastitis but at the cost of other efficiency gains, they focused on tracking, controlling and killing whichever germ was responsible for mastitis at that time, an agenda that was facilitated – but not driven – by drug company efforts to devise additional, more effective antibiotic preparations.

However, while the values and objectives of productivist agriculture certainly moulded mastitis research and control, they cannot fully account for it, because as recent studies have shown, there was no single, unidirectional route to greater farming productivity. In pig farming, for example, producers sought greater efficiency in a variety of ways, ranging

from high input–high output indoor systems of production, to low input–low output outdoor methods.[86] The analysis of scientists' unpublished discussions reveals that they too faced choices in how to investigate and control mastitis for the purpose of increasing milk production. Their ultimate focus on bacterial causes and standardized solutions was not inevitable.

Throughout the 1940s, Tom Dalling, head of the ARC mastitis conference, argued that mastitis research and control should pay more attention to the contributing roles of management, overstocking, driving cows, accidental injuries, soiled litter, poor hygiene and milking technique.[87] Alongside efforts to identify the optimal use of penicillin in mastitis control, he oversaw a number of farm surveys which aimed to correlate mastitis incidence with husbandry factors. The results – which were never published – were not clear cut. It transpired that the growing availability of penicillin left few herds in which the 'natural' course of mastitis could be studied, while ongoing changes in husbandry systems made it virtually impossible to isolate the effects of one management factor from the others. Due to these difficulties, the optimism surrounding penicillin, and the belief that without the germ there was no disease, this approach was abandoned.[88]

As already noted, in 1949 NIRD researchers planned to study the relationship between mastitis and management. However, they, too, found it difficult to draw meaningful deductions from the herd history of mastitis.[89] Discovering that mastitis was often absent from herds with defective milking machines, yet present in apparently well-run herds, they resisted its definition as a problem of management, not least because husbandry methods were in flux and good management meant different things to different people. They argued that while associations between mastitis and particular production practices were easily drawn, it was far harder to demonstrate causality. Only controlled experiments could reveal what factors were implicated, and which interventions worked. From their knowledge of mastitis epidemiology, they saw the milking machine and milking process as good places to start.[90]

At the CVL, scientists also showed interest in going beyond germs when considering the causes of mastitis, but their proposal to study the influence of nutrition was rejected on the grounds of cost.[91] The MMB, which promoted the use of the Bulk Milk Cell Count as a herd-level indicator of mastitis, attempted to relate the results to farm management, but the wider questions it raised about the influence of hormones, husbandry and inherited resistance fell beyond its remit to investigate as a near-market organization.[92] Various researchers suggested trying to breed mastitis-resistant cattle, but this was not pursued due to the lengthy, difficult nature of research, and concerns about a possible conflict with high yields.[93]

[86] A. Woods, 'Rethinking the history of modern agriculture: pig production in mid-twentieth century Britain', *Twentieth-Century British History,* 23 (2012), pp. 165–91; Theunissen, 'Breeding without Mendelism', Theunissen, 'Breeding for production'.

[87] TNA, JV 10/257, ARC committee on mastitis: Report for the period ended 31 Dec. 1940; Woods, 'Farm as clinic'.

[88] TNA MAF 189/358, MAF 189/436–7, Mastitis conference papers, 1944–49; S. Hignett, 'Farm health problems', *VR* 67 (1955), p. 900.

[89] J. Oliver, F. H. Dodd and F. K. Neave, 'Infection and mastitis in a dairy herd', *J. Dairy Res.,* 23 (1956), pp. 169–80.

[90] C. D. Wilson and F. H. Dodd, 'Mastitis', *VR* 75 (1963), pp. 1311–26; Dodd, Neave and Kingwill, 'Udder infection'.

[91] Wilson, 'Man, machines and mastitis'.

[92] TNA, JV 10/257, MMB report, 16 Nov. 1964.

[93] J. O. L King, 'Mastitis'; C. D. Wilson, 'Looking to the future in dairy science', *VR* 96 (1975), p. 22; Bramley and Dodd, 'Reviews'.

These examples show that, while enhanced milk production was an overarching goal of mastitis research, the decision to pursue it through germ-focused enquiries rather than wider frames of reference was based essentially on pragmatism. Located within organizations that were primarily concerned with near-market research, the causal roles of genetic and husbandry factors were simply too complex, too difficult and too costly for scientists to study. Investigating the bacterial causes of mastitis promised quicker and more commercially viable results.

Scientists' search for standard, universally applicable mastitis solutions was not an inevitable consequence of this approach. While they agreed that more intensive systems required more standard animals, they repeatedly acknowledged the individuality and diversity of cows, germs and stockmen. They often observed that there were multiple strains of *Staph. aureus*, whose different degrees of pathogenicity could affect the outcome of scientific trials.[94] Natural variation in cow susceptibility meant that they responded differently to hygienic and antibiotic interventions. Therefore the results of research in one herd could not necessarily be applied elsewhere, especially as scientists also differed in their methods, such that 'sampling procedure and timing, the design of trials, vested interests, and even optimism and pessimism are only a few of the factors which steer us towards results at variance'.[95] Cows also varied in their milking habits.[96] As C. D. Wilson pointed out in 1963, 'there is no such thing as a standard cow giving a standard volume of milk through a standard teat orifice': each milked at different rates. Leaving the machine on for a fixed amount of time might suit one cow, but could predispose others to mastitis by damaging their udders. The men who oversaw this process possessed varying degrees of an indefinable quality known as good stockmanship, which brought the 'human element' to bear on the incidence, spread and control of mastitis.[97]

Such variability and individuality contributed to the complexity of the mastitis problem. It led J. K. L. Pearson, a mastitis scientist working for the Northern Ireland government, to reject the philosophy of universal mastitis controls, in favour of individualized approaches that considered in turn the range of factors known to influence the disease.[98] However, while NIRD and CVL scientists agreed that 'understanding the causes [of difference] is probably the key to further improvements of the mastitis control system',[99] they chose not to investigate this matter. This was not simply because they thought that uniform controls would be more effective in promoting production, but because again, pragmatics won out. Instead of engaging with real-world complexity, they chose to simplify it, through a narrow focus on udders, germs and milking.

VI

In analysing the shifting identity of bovine mastitis, and scientists' multiple attempts to investigate and control it within the wider context of agricultural change, this article has illustrated the co-constitution of agricultural science and production. For the purposes of

[94] Wilson, 'Man, machines and mastitis'.

[95] J. K. L. Pearson and C. L. Wright, 'Dry cow therapy as a means of controlling bovine mastitis', *VR* 84 (1969), p. 297.

[96] F. H. Dodd, *Machine milking: the relationship between man, cow and machine* (1975).

[97] Wilson, 'Man, machines and mastitis', p. 1317.

[98] J. K. L Pearson *et al*., 'Factors involved in mastitis control', *VR* 91 (1972), pp. 615–24.

[99] Kingwill *et al*., 'Mastitis control system', p. 100.

mastitis control, milk production methods were scientized, while at the same time mastitis research was productivized. Influenced by the values and objectives of modern dairy farming, scientists constructed mastitis as a problem of production rather than health. Although mastitis prevalence remained high, their research did not fail because it facilitated the shift towards more intensive systems of milk production.

While important, productivism alone cannot explain the direction of scientists' research and their preferred mastitis solutions. Although their published papers give the impression that investigations and interventions unfolded in a logical, self-evident manner, scientists' unpublished and informal discussions show that there was no straightforward embrace of productivist agendas. Rather, scientists recognized multiple routes to productive efficiency, brought pragmatic considerations to bear on choosing between them, and acknowledged the tensions and problems associated with this choice. These findings add important nuances to the dynamic relationship between science and production. They help to explain how and why modern dairy production assumed the shape that it did, and the manner in which scientists contributed to this process.